A Nutritional Approach to Treating ADHD

Hyperactivity in children need not be treated with psychostimulant drugs like Ritalin, when nutritional approaches using essential fatty acids can produce beneficial results.

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AN OVERDIAGNOSED AND OVERPRESCRIBED DISORDER

Attention Deficit/Hyperactivity Disorder (ADHD) in children is fast becoming one of the most overdiagnosed and, many would argue, overprescribed childhood disorders both in the United States and now in Britain. It is certainly one of the most fiercely debated.

In the US its incidence is estimated at 3–5%, and up to 10% if less stringent criteria are used. In the UK it is put at up to 2% of children aged 6 to 16, with some 69,000 suffering "severe ADHD" (Baldwin S., *Crit. Pub. Health* 2000;10(4):453-62).

While the diagnostic criteria (see box) are disputed, the standard treatment—especially in the US—is even more controversial. Since the 1960s, the psychostimulant methylphenidate hydrochloride (MPH)—an amphetamine-like addictive drug that mimics the biochemical properties of cocaine—has been administered to thousands of children to the point where it is now estimated that up to one in seven American children may be given the substance daily.

A similar, staggering increase in use has been recorded in the UK, where MPH is designated a class B drug (class A if it is in solution). Professor Steve Baldwin at the University of Teeside (who, tragically, died in the Hatfield rail crash last year) stated that, from 6,000 a year in the UK in 1994, the number of prescriptions by 1997 had risen 15-fold to 92,000 (Baldwin, 2000, *op. cit.*). By 1999 this had reached 131,000 (covering some 21,000 children), but this is likely to be a gross underestimation because official statistics (based on pharmacy returns) do not include all prescriptions in private practices, young offender centres or residential homes.

In France, MPH use is rare, while in the rest of Europe its prescription for minors is uncommon or unknown. However, Baldwin and colleague Rebecca Anderson estimate that if MPH prescriptions were allowed to double year-on-year, by 2007 one in seven UK schoolchildren would be taking the drug daily (Baldwin and Anderson, *Crit. Pub. Health* 2000;10(1):81-6).

Indications that drug companies have this kind of scenario in mind is evidenced by the fact that since the end of 1999 the leading manufacturer Novartis (formally Ciba Geigy and Sandoz) has lost its sole product licence for MPH (as Ritalin), and others have launched their own brand (Equasym, Medeva) while three others (Mallinckrodt Inc., Schein Pharmaceuticals, MD Pharma) are preparing products (Baldwin and Anderson, 2000, op. cit.). However, their plans may be severely curtailed because of a number of high-profile lawsuits against Novartis (see box) and because of increasing public and professional awareness of the potentially damaging long-term effects and, in contrast, the growing evidence of the significant benefits of nutritional and other interventions.

THE PROMOTION OF MPH DRUGS

In the US, where Ritalin was first used in 1955, Novartis and other drug companies producing similar drugs used on children, such as dextroamphetamine and methamphetamine, have been very successful in persuading psychiatrists and health authorities of the alleged benefits of these drugs despite their potential risks and contraindications.

MPH is not licensed for children under the age of six (although it is used for those as young as three) or for children with marked anxiety, agitation or tension, symptoms or family history of tics or Tourette's syndrome, hyperthyroidism, severe angina or cardiac arrhythmia, glaucoma or thyrotoxicosis. Caution is required in the prescribing of MPH

US parents sue Novartis and APA for promoting Ritalin

Two lawsuits have been filed in California and New Jersey, asserting that Novartis, makers of Ritalin, and the American Psychiatric Association (APA) conspired to create a market for methylphenidate. These follow a class action launched in Texas in May 2000 by the Dallas law firm Waters and Kraus, alleging that since 1955 and through 1996 when it merged with Sandoz to become Novartis:

"Ciba/Novartis planned, conspired and colluded to create, develop and promote the diagnosis of Attention Deficit Disorder and Attention Deficit Hyperactivity Disorder in a highly successful effort to increase the market for its product Ritalin... It has repeatedly violated Article 10 of the United Nations Convention on Psychotropic Substances, 1019 UNTS 175 (1971)."

It continued:

"The American Psychiatric Association (APA) conspired, colluded and cooperated with the other Defendants while taking financial contributions from Ciba as well as other members of the pharmaceutical industry…"

Specifically, the company is accused of:

• actively promoting and supporting the concept that a significant percentage of children suffer from a "disease" which required narcotic treatment/therapy;

• actively promoting Ritalin as the "drug of choice" to treat children diagnosed with ADD and ADHD:

• actively supporting groups such as Defendant CHADD, both financially and with other means, so that such organisations would promote and support (as a supposed neutral party) the ever-increasing implementation of ADD/ADHD diagnoses as well as directly increasing Ritalin sales;

• distributing misleading sales and promotional literature to parents, schools and other interested persons in a successful effort to further increase the number of diagnoses and the number of persons prescribed Ritalin.'

Mr Richard Scruggs, one of the lawyers in the class actions, is quoted as saying that the Defendants "manufactured a disease. It has been grossly over-prescribed. It is a huge risk." (*BMJ*, 23 Sept 2000, p. 723)

The APA issued a statement last July, saying:

"Allegations that the [APA] conspired with others to create the diagnoses of [ADD and ADHD] as part of its *Diagnostic and Statistical Manual* so that medication could be used to treat these disorders are ludicrous and totally false. The APA will defend itself vigorously by presenting a mountain of scientific evidence to refute these meritless allegations, and we are confident that we will prevail."

The US support group CHADD (Children and Adults with Attention Deficit Disorder/Hyperactivity Disorder), which strongly advocates the use of Ritalin and is mainly funded by drug firms (it received $748,000 from Ciba/Novartis in the period 1991–94 alone), is accused of deliberately working to promote and increase the use of Ritalin, which has resulted in a huge increase in its use by children across the US, to the enormous profit of Ciba/Novartis. It is also accused of working to reduce or eliminate laws controlling the use of Ritalin in the US.

For further details, see the website www.ritalinfraud.com. [Also see "Class Action Lawsuit on Ritalin Fraud" in DeBriefings, NEXUS 7/06.]

for children and young people with epilepsy, psychotic disorders or a history of drug or alcohol dependence.

Proponents assert that MPH works by correcting a "brain disorder", "biochemical imbalance" or "biological dysfunction", but critics (Baldwin, 2000, op. cit.) assert that no scientific rationale for MPH prescription has ever been made explicit by its adherents (Jensen, P.S. et al., unpublished paper, Walter Reed Army Inst., Washington, 1989; Barkley, R.A. et al., *Pediatrics* 1989;86:184-92; Kewley, G., *BMJ* 1998;314:1594-5).

In November 1998, the US National Institutes of Health held a Consensus Development Conference on the diagnosis and treatment of ADHD. The 31 expert panel members (including Dr Breggin; see below) noted that no valid, reliable, independent test of ADHD exists and that there are "no data to indicate that ADHD is due to a brain malfunction" or that it might be a disease state or brain pathology (NIH, Rockville, 1998; see www.odp.od.nih.gov/consensus).

Despite this, supporters cite a large multimodal treatment study of ADHD, known as the MTA Study, which was sponsored by the National Institute of Mental Health at six separate sites (MTA Cooperative Group, *Arch. Gen. Psychiat.* 1999;56:1073-86). It compared four treatments, and proponents claim that it showed the superiority of stimulant treatment over behavioural and other treatments (although no nutritional alternative was tested).

However, one of America's fiercest critics of MPH, psychiatrist Dr Peter Breggin, of Johns Hopkins University's Education Faculty and author of *Talking Back to Ritalin* (Common Courage Press, 1998), has produced a 16-point critical rebuttal of the study that severely undermines its credibility and results (see www.breggin.com). Chief among his criticisms is that it was not a placebo-controlled, double-blind trial; the blind classroom raters found no difference in any of the treatment groups; there was no control group of untreated children; the children themselves did not rate themselves as improved; and out of 4,541 children originally screened, only 2.7% (123) completed the medication management trial.

His demolishing of the MTA Study is important. While the study lacks any evidence for MPH's efficacy, in Britain the National Institute for Clinical Excellence, which issued its Guidance on the use of MPH last October (NICE, 2000, "Technology Guidance No. 13"; see www.nice.org.uk), accepts in its assessment of any evidence the official MTA results with little critical analysis.

It refers to the study as "well conducted", when only one of the 125 listed references refers to a nutritional trial. It basically endorses the use of MPH, while acknowledging that "if improvement of symptoms is not observed after appropriate dose adjustment over one month, the drug should be discontinued". However, it does not mention that about 30% of children show no response to MPH or that up to 50% show side-effects. Baldwin (2000, op. cit.) states:

"Adverse drug reactions and side effects (more accurately described as 'main effects') from MPH include: CNS [central nervous system] sequelae, gastro-intestinal effects, cardiovascular effects, liver abnormalities, convulsions (including grand mal), drug dependency and addiction, drug withdrawal reaction, hair loss, low white blood cell count, agitation, hostility, depression, psychotic depression, abnormal thinking, hallucinations, psychoses, emotional lability, overdose and suicide [Breggin, *Ethical Hum. Sci. Services* 1999;1(1):13-33]. Paradoxically, the supposed desirable behavioural effects (including passivity, attention, reduced spontaneity) *are the primary toxic effects of psychostimulants*." [His italics]

The NICE Report mentions only nervousness and sleeplessness as common side-effects, and that other effects reported have been "relatively minor".

Despite the general acceptance by the US and UK psychiatric community to medicate this highly controversial disorder, there are now signs that the debate has reached a turning point with the launch of various legal actions in the US against not only Novartis but also the American Psychiatric Association for alleged fraud and corruption (see box, previous page), with similar actions apparently pending in the UK.

Now might seem an appropriate time for many MPH advocates to pause and consider non-drug treatments, specifically the growing evidence for the efficacy of nutritional and heavy metal detoxification treatments.

NUTRITIONAL APPROACHES AND INVESTIGATIONS

Essential fatty acid deficiency

Twenty years ago, Sally Bunday and her mother, Irene Colquhoun, founders of the Hyperactive Children's Support Group (HACSG) in the UK, were the first to propose that essential fatty acid (EFA) deficiency might be a factor in ADHD (Colquhoun, I., Bunday, S., *Med. Hypotheses* 1981;7:673-9). Surveying a group of hyperactive children, they found an excess of males, a link with asthma, eczema and other allergic conditions, and evidence from hair analysis of zinc deficiency. Clinical signs, such as excessive thirst, frequent urination, dry skin and dry hair, were observed that are consistent with EFA deficiency.

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In the United States, a diet developed by the late paediatrician Dr Benjamin Feingold (*Am. J. Nursing* 1975;75:797-803; *Why Your Child is Hyperactive*, Random, NY, 1975) was designed to eliminate certain synthetic additives and some foods, especially fruits, containing natural salicylates, which inhibit the conversion of long-chain polyunsaturated fatty acids to prostaglandins (see below). It was very successful in reducing symptoms, and groups sprang up all across the US and remain active in promoting and researching his treatment. In the UK, groups also started and the HACSG has adapted the diet for its own use.

Considerable evidence is accumulating that deficiencies in the body's reserve or production of EFAs is a major contributory factor in a range of interrelated childhood disorders, including ADHD, dyslexia, asthma, allergies and even autism, and that supplementation is valuable in a significant number of cases (Richardson, A.J., Ross, M.A., *Prostaglandins, Leucotrienes and Essential Fatty Acids* 2000;63(1-2):1-9). The clinical overlap between ADHD and, for example, dyslexia is around 30–50% in both directions.

Fatty acids play an essential role in brain structure and function. Two of them, arachidonic acid (AA) and docosahexanoic acid (DHA), play a major role in the brain and eye, constituting 20% of the dry weight of the brain and over 30% of the retina. Two others, eicosapentaenoic acid (EPA) and dihomogamma linolenic acid (DGLA), are crucial for normal brain development but play a more minor, structural role.

The absolutely essential fatty acids that cannot be synthesised by the body and therefore must be supplied in the diet are linoleic acid (n-6 series, to which DGLA and AA belong) and alpha-linolenic acid (n-3 series, to which EPA and DHA belong). Both AA and DHA are termed "longer-chain polyunsaturated fatty acids" (LC-PUFAs) and can usually be synthesised from their EFA precursors. The latter are critically important as precursors of a complex group of highly biologically active compounds including prostanoids (prostaglandins, thromboxanes and prostacyclins among others) and leucotrienes. These compounds perform numerous regulatory functions in the brain and the rest of the body.

Dr Alexandra Richardson (Physiology Lab, Oxford) and B. K. Puri (MRI Unit, Imperial College, London), in their important paper summarising the evidence ("The potential role of fatty acids in attention deficit/hyperactivity disorder", *PLEFA* 2000;63(1-2):79-87), state:

"EFA metabolism can influence many aspects of brain development, including neuronal migration, axonal and dendritic growth, and the creation, remodelling and pruning of synaptic connections [Crawford, M.A., in Bazan, N.G., ed., *Neurobiology of Essential Fatty Acids*, Plenum, NY, 1992:307-14].

Animal studies have shown that both neural integrity and function can be permanently disrupted by deficits of n-6 and n-3 fatty acids during foetal and neonatal development [Yamamoto, N. et al., *J. Lipid Res.* 1987;28:144-51; Neuringer, M. et al., *Ann. Rev. Nutr.* 1988;8:517-41; Bourre J.-M. et al., *J. Nutr.* 1989;119:1880-91].

"While both n-6 and n-3 fatty acids are required, the n-3 fatty acids such as DHA appear to play a special role in highly active sites such as synapses and photoreceptors, and deficiencies have particularly been linked to visual and cognitive deficits [Neuringer, N. et al., *J. Pediatr.* 1994;125:S39-47; *Proc. Natl Acad. Sci. USA* 1986;83:4021-5]."

Research by M. Makrides and co-workers has shown that infants may benefit considerably from the LC-PUFAs naturally present in breast milk but which are absent from many formula feeds (*Lancet* 1995;345;1463-8).

Although adequate supplies of EFAs are necessary throughout development and adult life to maintain normal function—and may be available—it is the conversion of the primary linoleic acid and alpha-linolenic acid into their LC-PUFA derivatives that is crucial for proper brain function. Unfortunately, a number of factors can interfere with the *conversion* of these parent EFAs to their respective LC-PUFAs, including:

- saturated or hydrogenated fats
- deficiency of vitamin and/or mineral co-factors (especially zinc deficiency)
- excessive alcohol
- stress hormones
- diabetes, eczema, asthma or other allergic conditions.

Thus, even if the diet contains sufficient EFAs, the child or adult may not receive adequate LC-PUFAs due to deficiencies in

Fluorescent Lighting can Stimulate Hyperactivity

The pioneering American photobiologist Dr John Ott drew on a range of plant, animal and human evidence in his classic work, *Health and Light* (Ariel Press, Columbus, 1973), to demonstrate how important natural light is for the health of the body and functioning of the brain, endorsing the inescapable fact that light is an essential nutrient.

Others, such as optometrist and light pioneer Dr Jacob Liberman, in his book *Light: Medicine of the Future* (Bear & Co, Santa Fe, New Mexico, 1991), and, in the UK, Dr Damien Downing (*Day Light Robbery*, Arrow Books, London, 1988; out of print), and most recently Dr Richard Hobday (*The Healing Sun*, Findhorn Press, 1999), have strongly supported this premise with a wealth of evidence and research.

By contrast, Dr Ott observed that the lack of the full spectrum of natural frequencies of light in many offices and classrooms had many adverse effects, including hyperactivity in children. Such "mal-illumination" was often caused by fluorescent lighting, which lacks the full spectrum and proper balance especially in the UV and blue/green frequencies. He reported that when fluorescent lighting was replaced by full-spectrum lighting (FSL) in classrooms, children's previous misbehaviour and hyperactivity were replaced by much calmer and more attentive behaviour. A study by M. Painter corroborated his observation and found a 32% drop in hyperactivity in children when fluorescent lights were removed from their classrooms (*Exceptional Children* 1981;47(5):352).

In 1973, a five-month study by Dr Ott's Environmental Health and Light Research Institute in Sarasota found (and filmed) dramatic change in hyperactive children. Under standard cool-white fluorescent light, children in two classrooms demonstrated nervous fatigue, irritability, lapses of attention and hyperactive behaviour. When these lights were replaced with FSL, marked improvement in behaviour began to appear, with children becoming calmer, more interested in their work and paying more attention. The results were published in a peer-reviewed journal, and similar results were obtained in experiments in two schools in California. Psychiatrist Dr Wayne London corroborated in a 1988 study in Vermont, which showed children stayed healthier during winter months, measured by a dramatic drop in absenteeism, if taught under FSL. Russian work reported by others has also confirmed that children exposed to FSL achieved higher marks, were less hyperactive and grew more quickly.

Interestingly, Dr Ott also reported that local dentists observed a 67% drop in cavities in children under similar conditions. Such findings were corroborated by a professor of dentistry at the University of Alberta, Canada, and it was also discovered that full-spectrum lighting even reversed the development of cavities (*New Scientist* 1991; 6 April, p. 13). Other studies have found that the number of cavities varied inversely with the amount of sunlight children were exposed to (*Am. J. Public Health* 1939;29:777; *J. Nutr.* 1938;15:547), an effect explained by the boosting of the photosynthesis of vitamin D (which requires UVB frequencies from the Sun), which is essential for the sufficient uptake of calcium to form strong bones and teeth.

To underline the stress that fluorescent lighting probably causes to young children's developing bodies and biological systems, the research by Professor Fritz Hollwich in Munich, as far back as 1980, should be brought to the attention of all teachers and parents of hyperactive children. Prof. Hollwich found significantly higher levels of the stress hormones cortisol and ACTH in those working under fluorescent lighting compared with FSL (*Ophthalmologica* 1980;180(4):188-97). His findings led the German government to ban the use of such lights in hospitals and medical facilities—an enlightened stand that has doubtless reduced the stress and improved the recovery of many patients, and which other governments and medical associations—not to mention all educational establishments—would do well to follow.

It is time that the above research received the attention it deserves.

conversion. In addition, individuals differ in their genetic constitutional ability to facilitate this conversion.

All the above, as well as disease factors, suggest the potential benefit of a dietary supplement of the preformed LC-PUFAs.

Clinical features suggestive of EFA deficiency

The higher ratio of boys to girls with ADHD is well accepted and varies from 2:1 to 10:1 (Szatmari, P. et al., *J. Child Psychol. Psychiatry* 1989;30:219-30). This is explicable using a fatty acid model, since males are more vulnerable than females to LC-PUFA deficiency (Huang, Y.S. et al., *Biochem. Arch.* 1990;6:47-54). The same level of male excess is also found in other developmental disorders that are clinically associated with ADHD, including dyslexia and dyspraxia (Stordy, B.J., *Am. J. Clin. Nutr.* 2000;71(supp. 1):3235-65; Richardson, A.J., Ross, M., 2000, op. cit.).

An excess of minor physical abnormalities is associated with ADHD (Quinn, P.O. et al., *Pediatrics* 1974;53:742-7) and EFAs, phospholipids and their metabolites play important roles in the cell abnormalities likely to underlie them (Hughes, D.A. et al., *J. Nutr.* 1996;126:603-10). Hyperactive children have also been found to have more chronic health problems, such as asthma or allergies, than normal children (Hartsough, C.S. et al., *Am. J. Orthopsychiatry* 1985;55:190-210).

Compared with normal children, ADHD children have been found to have a higher incidence of sleeping problems including difficulty settling, waking in the night and overtiredness in the morning (Trommer, B.L. et al., *Ann. Neurol.* 1988;24:325). PUFAs play a major role in the control of sleep mechanisms and directly affect the structure of neuronal membranes and indirectly affect the dynamics of complex lipids, prostaglandins, neurotransmitters, amino acids and interleukins that are required for the initiation and maintenance of normal sleep (Yehuda, S. et al., *Med. Hypotheses* 1998;50:139-45).

ADHD children exhibit more somatic complaints than normal children, including stomach aches, headaches, proneness to infections and general malaise with no obvious cause. In one study, 24% of ADHD boys and 35% of girls between 12 and 16 fulfilled the criteria for somatisation disorder (Szatmari, P. et al., 1989, op. cit.)

Because fatty acids and their derivatives play a critical role in regulating immune and digestive functions (Alexander, J.W., *Nutrition* 1998;14:627-33), EFA deficiency is known to contribute to general health problems such as proneness to infections and digestive and related disorders.

Symptoms of depression, anxiety and low self-esteem are typical in ADHD, whose co-morbidity with other behavioural and emotional disorders is common, with up to 44% having at least one other psychiatric disorder (Szatmari, P. et al., 1989, op. cit.). Increasing evidence is appearing that n-3 fatty acid deficiency may be important in depression (Hibbeln, J.R., *Lancet* 1998;351:1213; Peet, M. et al., *Biol. Psychiatry*

1998;43:315-19), and a recent double-blind, placebo-controlled study has shown the benefits of omega-3 fatty acids on the short-term course of illness in bipolar disorder (Stoll, A.I. et al., *Arch. Gen. Psychiatry* 1999;56:407-12).

Poor motor coordination is frequently observed in those with ADHD and, similarly, "soft" neurological signs such as motor overflow movements are also relatively common (Denckla, M.B. et al., *Arch. Neurol.* 1978;42:228-31).

Movement disorders in the general population are associated with deficiencies in LC-PUFAs (Nilsson, A. et al., *PLEFA* 1996;55:83-7) and thus poor motor coordination would be consistent with a lack of fatty acids.

ADHD's observed overlap with dyslexia (see above) appears to be stronger for attentional disorder without overt hyperactivity than for the mainly hyperkinetic form (Dykman, R.A., Ackerman, P.T., *J. Learn. Disabil.* 1991;24:96-103). The shared features include particular problems in specific aspects of visual and cognitive function (Conners, C.K., in Ravlidis, G., ed., *Perspectives on Dyslexia Vol. 1*, Wiley, Chichester, 1990:163-95). Deficiency in fatty acids has been proposed as contributing to dyslexia, and there is growing evidence that supplementation can help alleviate aspects of the disorder (Stordy, B.J., 2000, op. cit.).

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Evidence of EFA deficiency or abnormality in ADHD

In an early study, Michell and his colleagues found lower plasma levels of DGLA, AA and DHA in 44 ADHD children compared with 45 matched controls (*Clin. Pediatr.* 1987;26:406-11).

They also found that significantly more of 48 ADHD children compared with 49 age- and sex-matched controls suffered from polydypsia and polyuria as well as health problems and language, learning and reading difficulties.

More recently, studies at Purdue University have provided further confirmation of abnormal fatty acid metabolism in ADHD. A team led by Stevens (*Am. J. Clin. Nutr.* 1995;62:761-8) found that, compared with 43 normal controls, 53 ADHD boys:

- were less likely to have been breast-fed (breast milk contains the pre-formed LC-PUFAs such as AA and DHA, whereas most formulas do not);
- were more likely to suffer from allergies and other health problems (already known to be linked with EFA deficiency);
- showed clinical signs of EFA deficiency (excessive thirst, frequent urination, dry skin and hair, and soft or brittle nails);
- had reduced blood levels of certain LC-PUFAs (especially AA, EPA and DHA) but *not* their EFA precursors;
- had an adequate dietary intake of the EFA precursors.

The results support the hypothesis of EFA abnormalities in ADHD and confirm that the problem lies in the conversion of EFAs to LC-PUFAs. Some 40% of ADHD children had a raised frequency of clinical fatty acid deficiency signs compared with only 9% of controls.

Stevens and his team also showed that both clinical signs and blood biochemical indices of fatty acid deficiency were significantly associated with the severity of reported behavioural problems and the incidence of learning and health problems (Stevens, L.J. et al., *Physiol. Behav.* 1996;59:915-20).

Another team (Bekaroglu, M. et al., *J. Child Psychol. Psychiatry* 1996;37:225-7) has reported that the mean serum free fatty acid level in 48 ADHD children was significantly lower than in 45 matched controls. A further, significant correlation was found between zinc and free fatty acid levels in the ADHD children.

Early studies of GLA supplementation showed only equivocal or modest benefits (Arnold, L.E. et al., *Biol. Psychiatry* 1989;25:222-8) probably because, as Richardson and Puri suggest (*PLEFA* 2000, op. cit.), n-3 rather than n-6 fatty acid deficiency is more relevant in ADHD and because of the short treatment duration. Recent research indicates that LC-PUFA levels in the brain may take up to three months to recover from a chronic deficiency state (Bourre, J.-M. et al., *PLEFA* 1993;4:171-80), and this must been taken into account in future studies.

At a National Institutes of Health special workshop on omega-3 essential fatty acids and psychiatric disorders, held in Bethesda, Maryland, on September 2–3, 1998, J. R. Burgess (1998) from the Purdue team presented preliminary results of a double-blind trial with ADHD children with clinical signs of fatty acid deficiency. They found that supplementation with a combination of DHA, EPA, AA and DGLA (weighted in favour of the n-3 fatty acids) successfully changed the blood fatty acid profile of ADHD children, from which followed reductions in ADHD symptoms.

However, another double-blind trial showed no benefits from supplementing with pure DHA (Voight, R., NIH, Bethesda, 1998). Richardson and Puri (p. 84) suggest that one reason may be that DHA alone is ineffective and that other fatty acids, especially EPA, may account for the Purdue study's positive findings. They also point to the differences in subject selection; the Purdue study selected children based on prior indications of fatty acid deficiency, while no such pre-treatment indices were used in the other, which adopted very strict exclusion criteria, excluding any co-morbidity and ensuring that the sample consisted of children with "pure" ADHD diagnoses.

To investigate the importance of EPA, Richardson is currently involved in a study of the effects of supplementing ADHD children with Eye Q (www.equazen.com), a product that contains a 4:1 ratio of EPA to DGHA. Results may be available by the end of the year.

Zinc status and colourings/heavy metal toxicity

Complementing Richardson's research on direct nutrition, work by Dr Neil Ward, in the Chemistry Department at the University of Surrey and adviser to the HACSG, and others has emphasised the critical importance of maintaining adequate zinc levels and

reducing the ingestion of additives and the burden of heavy metals (e.g., lead, cadmium, mercury) in reducing ADHD symptoms.

Early work found that zinc deficiency caused hyperactivity syndrome in rats (*Pediatr. Res.* 1975;9:94-7). Further studies (*Arch. Gen. Psychiatr.* 1981;38:714-8; *J. Pediatr.* 1994;125:691-8) showed that additives such as tartrazine (E102), one of the 15 azo dyes permitted in food, can trigger hyperactive behaviour in some children. One double-blind, placebo-controlled study by Ward and co-workers showed that in hyperactive (HA) children tartrazine could induce a reduction in blood serum and saliva zinc levels, with an associated increase in urinary zinc output (*J. Nutr. Med.* 1990;10:415-31). This change was related to deterioration in behaviour and emotional expression.

Environmental factors, such as fluorescent lighting, should also first be eliminated.

In a further study of HA children, Ward confirmed that the former had statistically lower zinc and iron levels compared to controls for blood, urine and washed scalp hair (all $p < 0.001$) (*J. Nutr. Environ. Med.* 1997;7:333-42). HA children known to react to synthetic colouring showed a significant reduction in their blood serum zinc levels and an increase in urinary zinc output in response to ingesting either tartrazine or sunset yellow (E119). Many HA children also showed significantly high levels of aluminium, cadmium and/or lead in their urine or hair. Raised aluminium levels are associated with antisocial behaviour in children (*Biol. Trace Elem. Res.* 1986;11:5), while cadmium has an adverse effect on brain metabolism, particularly a depressive effect on levels of norepinephrine, serotonin and acetylcholine (Ward, 1990, op. cit.).

Moderate zinc deprivation in prepubertal monkeys has been found to adversely affect their performance in visual attention and short-term memory tasks, without affecting growth rate and without any overt signs of zinc deficiency (Golub et al., *Am. J. Clin. Nutr.* 1994;60:238-43). Zinc deficiency can cause a hyperadrenal condition (*Physiol. Behav.* 1979;22:211-5), and adrenergic and dopaminergic system dysfunction have been implicated in ADHD (Kaplan, H. et al., *Synopsis of Psychiatry*, 7th ed., Williams & Wilkins, Baltimore, pp. 1063-8). It may also be associated with a reduction in melatonin secretion (*Int. J. Neurosci.* 1990;52:239-41), which, in turn, would lead to a reduction in serotonin secretion, which is known to be linked to aggressive behaviour.

In his most recent paper (*Nutrition Practit.* 2000;2(2):43-5), Ward summarises the evidence for the benefits of diet and trace elements and reports a study of supplementation on ADHD children. Of those given either dietary modification involving elimination, trace elements (zinc, iron and selenium) or EFA, or a combination of trace elements and EFA, the "most dramatic improvement" occurred in those on the combination, in terms of blood serum and hair levels and reduction in behavioural problems over a 10-week period. In a further, as yet unpublished, study, he found a progressive decline in blood zinc levels in four children using Ritalin over a 12-month period.

Given that zinc is an essential co-factor in over 100 enzymes and particularly in the conversion of EFAs to LC-PUFAs, zinc status and appropriate supplementation, plus assessment of synthetic food additives and heavy metals, would seem of primary importance in treating ADHD children.

Diagnosing ADHD

ADHD is defined by the "core" signs of inattention, hyperactivity and impulsiveness, according to the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders* (*DSM*, 4th edition, 1994), and is known as "hyperkinetic disorder" in the 10th revision of the *International Classification of Diseases* (WHO, Geneva, 1992).

There are three subtypes of ADHD:

- combined type, with signs of inattention and hyperactivity/impulsivity;
- predominantly inattentive type, with inattention but not hyperactivity/impulsivity; and
- predominantly hyperactive/impulsive type, with hyperactivity/impulsivity but not inattention.

The diagnostic criteria further require that:

- the signs have persisted for at least six months to a degree that is maladaptive and inconsistent with the developmental level of the child;
- there must be clear evidence of clinically significant impairment in social or academic functioning;
- some impairment is present in two or more settings (usually at home and at school);
- some of the signs that caused impairment were present before the age of seven; and
- the signs do not occur exclusively during the course of a pervasive developmental disorder, schizophrenia or other psychotic disorder and are not better accounted for by other mental disorders (such as depression or anxiety).

The diagnosis of hyperkinetic disorder (HKD), sometimes used by UK clinicians, defines a subgroup of ADHD. HKD requires the presence of all three core signs: inattention, hyperactivity and impulsiveness. It also requires that all of the core symptoms were present before the age of seven years, are pervasive (present in two or more settings) and cause impairment. HKD is broadly similar to severe combined-type ADHD.

CONCLUSION

The work of researchers like Dr Alexandra Richardson and Dr Neil Ward point irrefutably to two prime causes of the symptoms associated with ADHD. Given the paucity of evidence for any long-term benefit from MPH, its primary toxic effects and the first report that early MPH treatment in minors does correlate with later stimulant abuse in adulthood (Lambert, N., Hartsough, C., *J. Learn. Disabil.* 1998;31:533-44), it seems only common sense first to assess the nutritional status and heavy metal burden of any presenting child, correct it and observe any improvements in behaviour before considering any highly potent drug therapy. Environmental factors, such as fluorescent lighting, should also first be eliminated.

Let the advocates of MPH remember Hippocrates' primary command: "First, do no harm." He might have added, "especially to children". ∞

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A Nutritional Approach to Treating ADHD

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Resources
ADHD Support Groups

United Kingdom

• **Hyperactive Children's Support Group (HACSG)**: The oldest support group in Britain, this voluntary group was founded in 1977 by the late Irene Colquhoun and her daughter Sally Bunday and registered as a charity in 1979. It is concerned with counselling, education and research and provides a quarterly journal to members. It takes a nutritional approach focused around elimination diets and the use of EFAs, and also advocates heavy metal assessment. It does not advocate the use of MHP. Free information is provided (send SAE to HACSG, 71 Whyke Lane, Chichester, W. Sussex PO19 2LD), and phone advice is offered between 10 am and 1 pm during weekdays on 01903 725182. Visit the HACSG website at www.hacsg.org.

• **Overload Network International**: This network is run by Janice Hill who provides advice (tel 0131 5554967) and information (send SAE to 58 North Fort Road, Edinburgh EH6 6HN). The Overload Network and **Attention Fife** (run by Barbara Naumann, tel 01592 890346) advocate a nutritional/detox approach, reject MHP and are calling for a public inquiry into a recent Scottish guidance committee's report advocating MHP.

• **ADHD National Alliance**: This new group is being forged by Jim Hedgeland of Contact a Family (170 Tottenham Court Road, London W1T 7HA, tel 0207 3801261) and is funded by the Department of Health. Its aim is to bring together the various approaches to ADHD, and it has a list of various other groups, from pro-medication to users of certain herbs and complementary therapies, which offer help/information on ADHD.

Australia

• Hyperactivity Association, 24/29 Bertram Street, Chatswood, NSW 2067
• Queensland HA Association, PO Box 107, Yeronga, Qld 4104
• Sue Dengate, Darwin ADD Support Group, PO Box 85, Parap, NT 0804
• Anne Swain, Allergy Service, Suite 210, RPAH Medical Centre, 100 Carillon Avenue, Newtown, NSW 2042

New Zealand

• Diane Wellacott, Auckland Hyperactivity Association, Box 51-675, Pakuranga, Auckland

USA

• Feingold Association of the United States, Box 6550, Alexandria, VA 22306; membership office: 127 East Main Street, Suite 106, Riverhead, NY 11901
• The Carl Pfeiffer Treatment Center, 1804 Center Point Drive, Suite 102, Naperville, IL 60563
• Dr Bernard Rimbaud, PhD, ARI, 4182 Adams Avenue, San Diego, CA 92116

About the Author:

Simon Best, MA, is a UK-based medical journalist and co-author of *Electromagnetic Man: Health and Hazard in the Electrical Environment* (Dent, London, 1989; St Martins Press, NY, 1989). He is also Editor of the *Electromagnetic Hazard & Therapy* news report, a newsletter which investigates EM fields, their health hazards and their positive applications in areas such as electrotherapy and magnetotherapy.

Simon's article, "Mobile Phones: Time to Take Precautions", was published in NEXUS 8/01.